DRUG TOXICITY AND SIDE EFFECTS IN LEPROSY.

[An ICMR Project]

A REPORT

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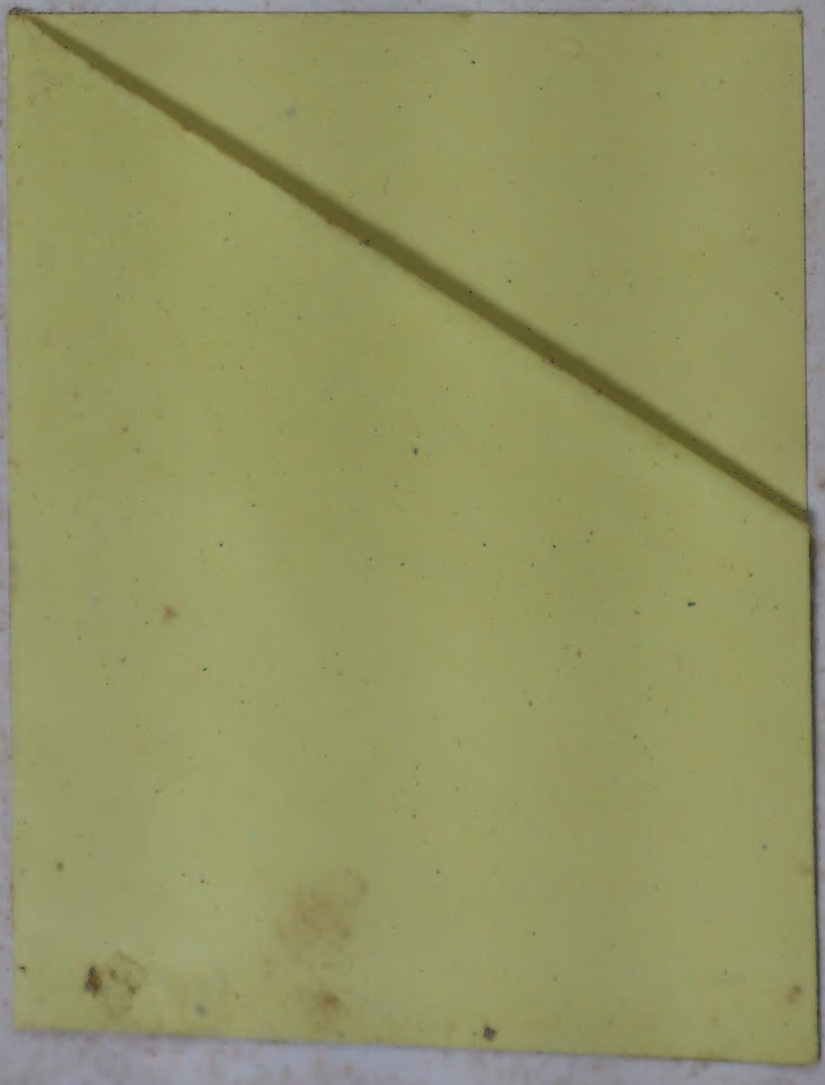

DRUG TOXICITY AND SIDE EFFECTS IN LEPROSY

(Indian Council of Medical Research Project)

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ABSTRACT

Retrospective and prospective studies were carried out to assess the neurotoxicity of Dapsone therapy in leprosy.

RETROSPECTIVE STUDY :

1. 1143 cases registered for treatment during the period between 1958-1984 were analysed. 118 patients had deformities (83 males and 35 females).

2. 69 cases developed deformities during treatment or added a new deformity to the already existing ones.

3. Of these, 46 cases (67%) had consumed 38 grams or less while 23 cases (33%) had consumed more than 38 grams of Dapsone.

4. Among cases who had consumed 38 grams or less 14 (20%) consumed the drug in one year while the remaining 32 consumed it over varying periods from 2-10 years. In other words the majority of deformed cases had irregular or/and insufficient treatment.

5. Acetylation pattern in those who did not develope deformities was similar to those who developed deformities.

PROSPECTIVE STUDY :

53 adult cases of TT and BT leprosy who showed thickening of a major nerve trunk with normal thickness of the same nerve on the contralateral side, were studied. These cases were randomly allocated a) to Dapsone in the dose of 100 mg/day (27 cases) and b) Rifampicin 300 mg/day (26 cases). Clinical and electrophysiological follow up in the form of nerve conduction velocity and electromyograph were carried out. 44 patients completed 3 or more follow up studies.

The motor conduction velocity showed no significant change initially or during follow up. Of the 44 patients 10 on Dapsone and 6 on Rifampicin showed evidence of peripheral sensory neuropathy initially and the remaining 28 did not show any NCV/EMG abnormality.

Of the 28 patients who had normal initial study, temporary sensory deterioration was observed in one case but improved with further treatment. Of the 16 patients who had shown evidence of peripheral neuropathy on initial study in 14 cases the deteioration remained stationary or improved with treatment and continuation of treatment did not cause any further damage. In these cases it was obvious that the disease process was arrested with treatment and no further damage to nerves occured.

Two patients (one on Rifampicin and one on Dapsone) continued to deteriorate while on treatment. However, while clinical improvement was marked, the electrophysiological studies showed deterioration. These two cases were clinically inactive. There was no fresh development of peripheral anesthesia, motor deficit or acute neuritis during treatment.

Disease per se arrested by the drug but leading to fibrosis or due to the poor regeneration of myelin in the diseased portion of the nerve might be the reason for the deterioration observed.

ANIMAL EXPERIMENTS :

To find out a direct evidence, nerve conduction studies are being carried out in dogs fed with increasing doses of Dapsone. There is a trend of decrease in motor nerve function in dog receiving Dapsone five times that of human dose. It is premature to comment further at this stage.

INTRODUCTION

Leprosy is caused by Mycobacterium leprae. It mainly affects the nerves and skin. Involvement of the nerves results in structural and functional damage. This is exhibited as disability or deformity. If leprosy did not cause deformities or disabilities it would not have been a dreaded disease. It would have been considered as just another skin disease. As at present, the disease is usually equated with deformity. The strong emotional bias against the disease in the society at large is mainly due to the disfigurement caused. Deformities are produced by the progress of the disease or due to neglect of the patient to take care of himself.

At present 70% of the Leprosy patients are not suffering from any significant deformities. The deformities are dependent on the involvement of many mixed nerves. It would be reasonable to expect that early treatment before the disease has spread and involved major nerve trunks should prevent deformities. This is debatable because it is not certain how effective the treatment is in preventing deformities when started after the nerves have been involved.

1908 fromm and wallmann Streptococcus TB

Sulphones were first successfully tried against leprosy in 1941 by faget. Diamino - diphenyl sulphone (Dapsone) was considered toxic and as such its derivatives namely Promine, Sulphetrone and Diasone were the early group of sulphones used against leprosy. It was soon found, however, that in the human system these derivatives were re-converted to Dapsone. It was, therefore, logical to use the parent substance itself, although in much smaller doses. Cochrane (1947) undertook trial with Dapsone and confirmed the therapeutic effect of the drug in low doses. Subsequently, Dapsone has been used in mass therapy of leprosy during the past 40 years routinely in leprosy control programmes.

Apart from its use in leprosy, Dapsone was found to be effective against other conditions like malaria, psoriasis etc., In these conditions the drug was used in doses ranging upto 1200 mgs a day. Under these high doses, neuropathy as a result of Dapsone toxicity was observed and a few case reports of such Dapsone neuropathy appeared from different centres. (Allday and Bernes 1951, Fredericks et al, 1976, Williams 1972, Epstein et al 1976, Voliden 1977, Gehrmen et al 1977, Malkinson and Pearbon 1977, Prayag et al 1979, Homedia et al 1980, Zorhar Argov et al 1979, Warren 1980) It is

to be noted that all the authors used Dapsone at a dose much higher than the dose used at present against leprosy.

Desikan (1968) on the other hand, was of the opinion that Dapsone could prevent the onset of deformities by its neuroprotective action.

A few workers from India drew attention to the possibility of neurotoxicity of Dapsone in leprosy patients. Noordeen & Srinivasan (1966) reported relatively low disability rate among those who are not taking treatment. Comparing the disability rates among treated and the untreated they found that among the treated 45.6% and among the untreated only 9.8% had disability. They suggested that treatment for leprosy, given under field conditions could possibly result in increased disability in the patient population.

Gupte (1979) showed statistically that patients who are receiving dapsone therapy in large amounts and regularly are more often deformed than the patients who are irregular and receiving lesser amounts. These reports naturally caused an alarm since Dapsone is the sheet anchor of leprosy treatment and has been extensively used in mass therapy and control programmes.

Experimental studies in mice by Kamala et al (1984) did not support the hypoothesis that Dapsone produced neurotoxicity. Their work actually indicated that Dapsone is neuroprotective than neurotoxic in leprosy.

Since the reports of Noordeen & Srinivasan (1966) and of Gupte (1979) were based on retrospective statistical analysis, it was felt that a prospective study should be planned to investigate the possible neurotoxicity of Dapsone. Such a work has been undertaken. Simultaneously a retrospective study of case records was also conducted from one of the centres where treatment of leprosy routinely with Dapsone was carried out for well over 30 years.

RETROSPECTIVE STUDY

The retrospective study is based on the analysis of the records of Sevagram Control Unit run by Gandhi Memorial Leprosy Foundation. This unit was started in 1952 when the prevalence of leprosy was 25.7/1000 pop. At the time of this study the leprosy prevalence came down to 6.3/1000 pop.

The records of Sevagram Control Unit were analysed and only those patients who had developed deformity during the course of treatment were selected out. The mean drug consumption in a 5 year interval was calculated both for the deformed group and the undeformed group. The undeformed group of patients also consumed Dapsone but did not develop any deformity during the treatment period. The amount of Dapsone obtained was compared in both the groups.

OBSERVATIONS AND DISCUSSION:

Retrospective analysis of the patients records from 1958-84 was carried out. 1143 patients received treatment during 1958-84,out of which 113 patients were deformed. Broadly the total number of patients were divided into 2 groups.

1. Deformed group - 118 (10.3%)
2. Undeformed group - 1025 (89.7%)

Total Number of patients - 1143 (100%)

There were 587 (57.3%) males and 458 (42.7%) females in the undeformed group and 82 (69.5%) males and 36 (30.5%) females in the deformed group.

It was observed that the maximum number of cases belonged to tuberculoid and maculo anaesthetic types of leprosy both in deformed and non-deformed groups.

The total number of patients deformed 118

1. Undeformed patients who developed a new deformity during the course of treatment 51
2. Patients who were deformed but not showed any additional deformity during the treatment period 49
3. Deformed patients who developed an additional deformity during the treatment period 18

The nerve involvement at the time of initiation of treatment can be classified as

N1 = Not thickened nerve

N2 = Palpable thickened nerve without tenderness

N3 = Palpable thickened nerve with tenderness

N4 = Thickened or fibrosed nerve associated with deformity/ies.

(see Table I)

Table I : Majority of the patients in not deformed group belonged to N1 category. The deformed group belong to all categories equally. In the N4 group 18 (26.1%) patients showed a further increased or appearance of new deformity to the already existing one. The difference between this group and those not developed deformity under treatment is statistically significant (Z=4). The N3 group of deformed patients (16) when compared with undeformed group showed a higher risk of development of deformity (Z=3.5). Therefore, it can be concluded that N3 group and N4 group of patients are more likely to develope a fresh or added deformity during the course of treatment.

Most of the patients 959 (83.8%) reported to the clinic or detected by survey within 12 months of noticing the symptoms of the disease. This is true for both the deformed and not deformed groups. In other words the deformity status has no relation with the duration of the disease process.

Drug collection can be classified as follows :

Category	I	< 25% of the due drug collection
	II	25 - 49% of due drug collection
	III	50 - 74% of due drug collection
	IV	> 75% of due drug collection

(see Table II)

Table II: It can be noticed that out of 309 patients who received >75% of drug, 32 (10.4%) developed deformities and the rest 277 (89.6%) escaped. Out of 384 patients who received <50% of drug, less number of patients 12 (6.6%) developed deformity. The statistical significance (X test) showed that the difference between category III and IV and II and III are significant. This concludes that as the patients are more regular, the risk of development of deformity increases.

Table III,IV,V & VI: It was observed that the dose of Dapsone was not constant. Initially very low doses of Dapsone were prescribed. From 1978 onwards, the dose of Dapsone was increased to the usual therapeutic dose of 100 mg. So the total number of patients were divided into 2 groups.

Group A - Who received minimal doses of Dapsone till 1978.

Group B - Who received the therapeutic dose of Dapsone (100 mg) from the beginning from 1978-84.

Group A(Tables III & IV): The mean quantity of drug consumption of these patients was calculated and compared with the duration of its consumption. It was observed that there was no change in the mean drug consumption in both deformed and not deformed groups. There was no statistically significant difference in both the groups.

Group B (Tables V & VI) : In 2-5 years duration the mean drug consumption by non deformed group was 43.14 Grams. (SD + 16.76) and by deformed group it was 68.96 Grams. (SD + 38.24). Though apparently there appears to be a difference, this is not statistically significant. (Z = 1.16 at 95% confidence limits)

There is a statistical significant difference in the amount of drug consumption in the deformed group,(115.7 Grams. (SD + 43.34)) and not deformed group (65.06 Grams. (SD + 43.36)) in 6-10 years duration. Here it is worth mentioning that the total drug consumption till cure was calculated in both the groups. To be precise the amount of Dapsone consumption till the development of deformity should be calculated in the deformed group. This amount should be compared with the drug consumption in the not deformed group within the same period as deformity group. This will be able to establish an association of Dapsone and the development of deformity but such information could not be ellicited from the records.

Grouping of various frequencies was done to facilitate the comparison.

N1 + N2 Group I

N3 Group II

N4 Group III

Even the drug collection was also clubbed to form 3 groups.

Group 1 < 38 Grams of Dapsone

Group 2 39-76 Grams of Dapsone

Group 3 > 77 Grams of Dapsone.

(see Table VII)

This table indicates how much drug had been consumed by the patient before the development of deformity. It was observed that though the drug consumption was < 38 Grams, 14 (20.3%) developed deformity within one year of treatment duration. On the other hand 29 (42.0%) developed deformity consuming the same amount but spread over a span of 2-5 years.

It is striking that the deformities developed within 0-5 years (54 out of 69) duration. So even though the patient continues to consume Dapsone after 5 years, there are less chances of development of deformity.

It is clear that there are some factors responsible for development of deformity other than Dapsone consumption.

These can be

1. Initial nerve thickening
2. Acetylation pattern
3. Reactions during treatment.

(see Table VIII)

Table VI I : This table shows the acetylation status of 22/69 patients who developed deformity and alive but not receiving Dapsone any further because they had been cured of leprosy. Acetylation status of 22 controls was also determined. It can be observed that the deformed cases and matched controls showed similar distribution and frequencies. These were not statistically significant (x=0.51). This suggests that cases and controls are at par and their acetylation status may not play any role in the causation of deformities.

It was observed that 17 out of 33 in deformed group experienced reactions more than once. But in the not deformed group 34 out of 80 had more than 1 reactive episode. Severe reactions were common in not-deformed group (19 out of 70) when compared with those in deformed group (2 out of 33)

The not deformed group experienced reactions (61 out of 1074) i.e. 5.6% had mild reactions. The deformed group experienced reactions (31 out of 69) i.e. 44.9%.Statistically these values were highly significant (z = 7.2) But difference in the frequency of severe reactions is not significant. In other words,the deformed group experienced more mild reactions than the not deformed group. It is apparent that the initial nerve involvement and the frequency of mild reactions play a major role in causation of deformities in leprosy patients.

TABLE I

Distribution of cases according to Nerve Involvement

Type of Nerve involvement	Deformed during treatment		Not deformed during treatment		Total	
	No.	%	No.	%	No.	%
N1	19	27.5	690	64.2	709	62.0
N2	16	23.2	316	29.4	332	29.0
N3	16	23.2	19	1.8	35	3.0
N4	18	26.0	49	4.5	67	5.8
TOTAL	69	99.9	1074	99.9	1143	99.8

TABLE II

Distribution of cases according to treatment category

Percentage of Drug collection	Deformed during treatment		Not deformed during treatment		Total	
	No.	%	No.	%	No.	%
< 25%	3	3.6	82	96.4	85	7.4
25 - 49 %	9	3.0	290	97.0	299	26.1
50 - 74 %	25	5.5	425	94.5	450	39.2
> 75%	32	10.4	277	89.6	309	27.1
TOTAL	69	6.03	1074	93.97	1143	99.8

TABLE III

Distribution of cases according to mean consumption of drug and duration of treatment in the non-deformed group (1958-1978)

Duration of treatment	No of patient	Total Drug consumption	Mean (Grams)	SD (Grams)
< 1 yr	30	46.72	1.557	1.90
2-5 yrs	400	9313.4	21.66	19.20
6-10 yrs	155	6639.3	42.83	39.98
11-15 yrs	5	420.7	84.14	41.68
16-20 yrs	1	217.2	217.2	
Total	631	16637.3	26.3	

TABLE IV

Distribution of cases according to mean drug consumption and duration of treatment in the deformed group (1958-1978)

Duration of treatment	No. of patients	Total drug consumption	Mean (Grams)	SD (Grams)
< 1	-	-	-	-
2-5	8	206.3	25.78	13.36
6-10	24	1241.0	51.70	20.20
11-15	11	747.0	67.99	36.35
16-20	2	179.5	89.75	67.81
TOTAL	45	2373.8	52.75	

TABLE V

Distribution of cases according to mean consumption of drug and duration of treatment in non-deformed group (1979-84)

Duration of Treatment	No.of patients	Drug consumed (Grams)	Mean Drug consumption (Grams)	SD
< 1	65	341.47	5.253	4.41
2-5	306	13301.55	43.142	16.76
6-10	53	3448.59	65.067	43.36
11-15	8	795.30	99.412	50.13
16-20	6	799.48	133.246	52.68
21-25	5	829.16	165.8	46.75
TOTAL	443	19515.55	44.05	-

TABLE VI

Distribution of cases according to mean consumption of drug and duration of treatment in the deformed group (1979-84)

Duration of treatment	No.of patients	Drug consumed (Grams)	Mean drug consumption	SD (Grams)
< 1	-	-	-	-
2-5	3	260.9	68.966	38.34
6-10	4	462.8	115.7	43.34
11-15	1	117.5	117.5	-
16-20	7	1250.8	178.685	52.08
21-25	9	1850.5	205.616	72.47
TOTAL	24	3888.5	162.02	

TABLE VII

Distribution of cases according to years of development of fresh/added deformity and the amount of Drug consumed in that period in the Deformed group under treatment

Time taken to develop deformity in years	Drug consumed during the period <38 Grams	39-76 Grams	>77 Grams	Total
< 1 year	14(20.3%)	-	-	14(20.3%)
2-5 years	29(42.0%)	9(13.0%)	2(2.8%)	40(58.0%)
6-10 years	3(4.3%)	8(11.5%)	1(1.4%)	12(17.4%)
11-15 years	-	2(2.8%)	1(1.4%)	3(4.3%)
16-20 years	-	-	-	-
21-25 years	-	-	-	-
TOTAL	46(66.6%)	19(27.5%)	4(5.8%)	69(100%)

TABLE VIII

Distribution of cases and controls according to acetylation status of Dapsone

Acetylation status	Deformed Cases		Controls		Total	
	No	%	No	%	No	%
Rapid	4	18.2	6	27.3	10	22.77
Slow	18	81.8	16	72.7	34	77.23
TOTAL	22	100.0	22	100.0	44	100.0

PROSPECTIVE STUDY

A prospective study was planned to investigate the possible effect of Dapsone therapy among patients attending the leprosy clinic at the Civil Hospital, Wardha.

PATIENTS AND METHODS

Paucibacillary type of leprosy patients belonging to T T and B T types were selected for the study. Multibacillary types were avoided on ethical consideration, since the study involes monotherapy. Patients fulfilling the following criteria were selected for the prospective study :

1. Age of the patient should be 15-40 years.
2. Atleast one nerve (either ulnar or lateral popliteal) should be clinically thickened and the contraleteral nerves should not show any clinical thickening. The criteria was laid down so that the same patient could serve as a test case and could also be his own control.

3. The cases should preferably be untreated. However, patients with a history of having taken treatment for a period not exceeding 3 months may also be included if untreated cases are not available.
4. They should not have diabetes or be alcoholics. Patients with any other concommitent cause of neuropathy should also be excluded.

It was proposed to include 50 paucibacillary patients. All the patients were thoroughly examined in the clinic in good day light. The skin lesions were carefully recorded on a body chart. The sensations over the patches and over the extremities were carefully tested and mapped in the body and extremities charts. All the peripheral nerves were carefully palpated and the following characteristics were particularly noted -

(a) shape of the thickening, (b) extent or the thickening, (c) presence or absence or tenderness.

The functional assessment of muscles supplied by the nerves was done and positive findings if any were noted. Skin smears were obtained from the standard and selected sites and examined. Lepromin test was also performed, noting the early as well as late reactions. A skin biopsy was obtained from a typical skin lesion. The diagnosis was thus made on the basis of clinical findings, lepromin readings and histopathology.

The following investigations were also conducted on the day of registration.

Blood :

Haemoglobin in grams

Total and differential count

Serum proteins

SGOT

SGPT

Blood sugar

Urine :

Albumin

Sugar

Microscopy

Acetylation studies of Sulphadimidine.

Nerve conduction studies (Motor and Sensory nerve conductions) and also Electromyographic studies were conducted during the week of registration. These studies were carried out by Dr. G.M. Taori at the Central India Institute of Medical Sciences, Nagpur. MS 4 Electromyographic machine was used. The electro-physiological studies were conducted using standard techniques. The studies were conducted in an air conditioned room keeping the temperature nearly constant for all patients throughout the study.

The patients were administered Dapsone (100 mgm) or Rifampicin (300 mgm). While the treating physician knew which patient received Dapsone or Rifampicin, the neurophysiologist was unaware of the type of treatment given. The patient attended the clinic once in two weeks. The patient consumed one dose in the presence of the physician and remaining 13 doses were self administered. The regularity was maintained with the full cooperation of the patients. Home visits were made whenever required.

Clinical examination was repeated every 3 months. All the laboratory investigations and electrophysiological studies were repeated every six months. At the end of two years the patient was declared cured if he showed no signs of activity. The treatment was then stopped.

All the patients were advised to report to the doctor at any time if they had any complaints regarding their condition. The drug intake was ensured by surprise home visits and by counting the tablets and urine examination. The urine was checked by its colour for Rifampicin intake or by the spot test for Dapsone intake. A check was also made to see that the patient did not receive any drug which can potentiate neurotoxic effect.

All the patients tolerated the drugs well. There were no adverse effects either for Dapsone or Rifampicin. All the patients had more than 90% attendance.

OBSERVATIONS AND DISCUSSION

1. Age and sex distribution of cases

The findings on a total of 53 patients are presented. Of these, 27 belong to Group I and 26 belong to Group II. There were 38 males and 15 females. Since all the patients are adults, the doses are fixed and uniform. The following table shows the age sex distribution of the patients. (see Table I)

TABLE I: Maximum number of patients 18 (33.9%) belonged to 20-24 year group followed by 15-19 years (12 patients 22.6%) and 25-29 years group (12 patients 22. There were 10 patients (18.8%) in the age group 35-45 years. The male:female ratio is 2.5:1. The age distribution and sex ratio are similar to the prevalent age and sex distribution of leprosy patients in Wardha district.

2. Duration of Disease

Table II shows the mean duration of leprosy patients in both groups.

TABLE II: The 27 patients in Dapsone group had a mean duration of disease 13.214 months. The Rifampicin group of patients had a mean duration of 9.85 months. It was found by the 't' test that the difference is not significant.

3. Acetylation pattern

Table III shows the acetylation pattern in the two groups of patients.

TABLE III :The acetylation pattern was studied with sulphodimidine in both the groups. All the patients could not be studied due to certain operational problems. 17 patients from Dapsone group and 15 patients from Rifampicin group could be stulied for their acetylation pattern. It is seen that out of 17 patients who are receiving Dapsone, 13 patients (76.5%) are slow acetylators and 4 (23.5%) are rapid acetylators. Out of 15 patients who are in Rifampicin group 11 patients (73.3%) are slow acetylators and 4 (26.5%) are rapid acetylators. This characteristic of rapid or slow acetylation coincides well with the figures obtained for

leprosy patients in Wardha as confirmed by random studies in 22 other cases attending the clinic. Furtherr the Dapsone and Rifampicin groups show comparable acetylation pattern.

4. Involvement of Nerves

Table IV shows the involvement of nerves.

TABLE IV: In a total of 53 patients 40 ulnar nerve thickening and 14 lateral popliteal nerve thickening were found. This would result in a total of 40 affected ulnars and 40 healthy ulnars and 14 affected lateral popliteals 14 healthy lateral popliteal nerves, which were studied clinically and electrophysiologically.

5. Electro-physiological findings

As already mentioned, electro-physiological studies were conducted in all the patients once every six months. Four observations were completed in about half the number and in the remaining half 3 or less examinations proposed were completed. The number of examinations on the cases are detailed in Table V.

Details of electro-physiological investigations in the groups of cases examined once, twice, thrice and four times are given in Tables VI, VII, VIII and IX.

6. Clinical findings

For further analysis of neurological changes only patients who had 3 or more studies have been included. Total number of such patients is 44. The reason for analysing only these patients is that the studies are near completion or completed.

For the 44 cases examined 3 or 4 times. Initial and final clinical findings are detailed in Table X along with the initial and final status of sensory nerve conduction studies.

TABLE X: A clinical assessment of the 44 cases may first be noted. There were 22 patients receiving Dapsone and 22 patients receiving Rifampicin. All the patients who were on Dapsone showed clinical improvement comparable to that of Rifampicin Group.

In 21 cases on Dapsone, the hypopigmented patches became faint and in 1 case, patches completely disappeared within 2 years of treatment.

Motor functional deficit was observed initially in 4 cases of affected lateral popliteal nerves and in 5 cases of affected ulnar nerves. The motor functional deficit was associated with peripheral anaesthesia in 3 out of 9 cases and only in one case there was motor deficit without peripheral anesthesia. All these 9 nerves showed improvement within 3-6 months of treatment

irrespective of drug (Rifampicin or Dapsone).

There were 8 patients initially (4 receiving Dapsone and 4 receiving Rifampicin) who showed thickening and tenderness of nerves. Of these only 2 nerves showed abnormal sensory conduction velocity. After 2 years of treatment, these two abnormal readings improved and became normal.

7. Correlation between clinical and electrophysiological findings:

Out of the 44 cases who had 3 or more examinations, 32 cases had thickened ulnar nerves and 12 had thickened lateral popliteal nerves. The sensory conduction studies were carried out in 32 affected ulnar nerves and the 32 contralateral clinically unaffected nerves. The findings are summarised in Table XI.

TABLE XI: There was no anesthesia in any of the 32 limbs with clinically unaffected ulnar nerves. In the limbs with affected ulnar nerves, anesthesia was present in 5 limbs and absent in 27 limbs. In the 5 cases with anesthesia, 2 showed abnormal sensory conduction latency and amplitude. In the 59 limbs without anesthesia, it is interesting to note that there were abnormal findings in

8 limbs, 5 of which had diminished amplitude, and 3 had diminished amplitude and increased sensory latency. This shows that the sensory conduction abnormality would be detected before clinically detectable anesthesia. Further, changes in the amplitude appears to be a more sensitive finding.

Correlation between the number of patches and sensory conduction is given in Table XII.

TABLE XII: Except for 4 cases all the rest had only 1-3 patches. As such no comparative analysis can be made according to the number of patches.

It was seen in Table V that out of a total of 44 cases examined 3 or 4 times, 16 cases showed abnormal electro-physiological findings. The duration of the disease in the cases with normal and abnormal initial findings is given below.

	Normal NCV,EMG	Abnormal NCV,EMG
Mean duration in months	12.314	14.588

Since the duration of the disease is more or less equal in both the groups no conclusions can be drawn.

Table XIII shows the initial and final mean motor and sensory nerve conduction velocities of the Dapsone and Rifampicin groups.

TABLE XIII a, b and c: Looking at the motor nerve conduction velocity of the ulnar and lateral popliteal nerves, it is seen that the conduction velocities of the affected and healthy nerves of both Dapsone and Rifampicin groups show no significant difference as confirmed by student 't' test.

We would have expected a significant difference if there had been a gross nerve destruction as a result of either the disease process or Dapsone toxicity. The lack of any detectable difference in motor nerve conduction velocity may be explained as follows :

a) In case of leprosy the nerve thickening is due to interneural, intraneural or peri-neural oedema. This oedema may not be causing any interference in the nerve conduction velocity. In other words there is no nerve damage.

b) The damage to nerves, if any, may be so minimal that it is not detectable by motor nerve conduction velocity studies.

c) Leprosy might be affecting one type of nerve fibres eg., slow conduction fibres. Since the nerve as a whole is being studied a difference may not be found. If individual nerve fibres are studied, they may be showing the change.

d) The fast conducting and slow [illegible] might have been damaged simultaneously but, the overall nerve conduction may not be showing any change because of compensatory mechanism.

e) Further, the normal values of motor nerve conduction of the ulnar and lateral popliteal nerves show a wide range (ulnar 40-60 metres per second; lateral popliteal 43-57 metres per second). As such any small difference within the range is not significant.

For the same reasons given above it is also seen that there is no difference in the Distal latency between affected and healthy nerves both initially and at the final recording in the Dapsone as well as Rifampicin groups.

On the other hand, it is seen from Table XIII that the sensory amplitude and sensory latency show a significant difference between healthy and affected nerves. As already indicated in the discussion on Table XI, sensory conduction is more sensitive as measured by amplitude and latency.

Summarising the findings in Table XIII, the present study does not show any difference in the initial and final recordings of motor and sensory nerve conduction velocities. This is because these are the aggregate values in a limited number of subjects. However, certain distinct changes are seen in individual cases and they are detailed below.

Of the 44 patients 16 showed evidence of peripheral neuropathy initially and the remaining 28 did not show any NCV/EMG abnormality. Out of these 16 cases which showed peripheral neuropathy initially, 10 belongs to Dapsone group and 6 to Rifampicin group.

Of the 28 patients who had normal initial study only one case on Rifampicin with ulnar nerve thickening showed deterioration during the follow up while on treatment. The deterioration noted was in the second study after 6 months of treatment. No further deterioration occured with continued treatment. The neuropathy may possibly be due to the progress of disease process which was arrested with treatment.

Of the 16 patients who had shown evidence of peripheral neuropathy on initial study 9 showed improvement and 3 remained nearly unchanged. Of these 12 cases, 8 were on Dapsone and 4 on Rifampicin therapy. It is obvious that the disease process was arrested with treatment and no further damage to nerves occured.

The remaining 4 cases showed further deterioration on continuation of treatment. It was found that 2 received Dapsone and 2 received Rifampicin. One case on Dapsone and one on Rifampicin therapy showed no further deterioration on the third reading. It is likely that in these 2 cases also the nerve conduction abnormality was

due to disease process, the progress of which was arrested by 6 months of treatment.

Two patients (one on Rifampicin and one on Dapsone) continued to deteriorate while on treatment. However, while clinical improvement was marked, the electrophysiological studies showed deterioration. These two cases were clinically inactive. There was no fresh development of peripheral anesthesia, motor deficit or acute neuritis during treatment. The following reasons could be attributed to the electro-physiological deterioration.

a) Disease could be unresponsive to the drug.

b) The deterioration could be related to drug concentration in the diseased nerve.

These two reasons can be ruled out as the patients responded well to the drugs (Dapsone and Rifampicin) and there was no clinical deterioration.

c) Disease per se arrested by the drug but leading to fibrosis or due to the poor regeneration of myelin in the diseased portion of the nerve. This can be the most likely reason for the deterioration observed in electro-physiological studies.

Table I

Age, Sex distribution of study and control groups

: in Years	MALE		FEMALE		Total
	DDS group	RFP group	DDS group	RFP group	
-19	3	4	1	4	12(22.6%)
-24	11	5	-	2	18(33.9%)
5-29	4	3	2	3	12(22.6%)
0-34	-	1	-	-	1(2.0%)
5-40	4	3	2	1	10(18.8%)
	22	16	5	10	53(100.0%)

Table II

Duration of Disease

Drug	Duration of Disease in Months		
	Male	Female	Mean
Dapsone(27)	13.772	11.16	13.214
Rifampicin(26)	10.875	8.25	9.86

Table III

Acetylation status of leprosy patients

	Acetylation status		Total
	Slow	Rapid	
1. Dapsone group	13	4	17
2. Rifampicin group	11	4	15
Total	24	8	32

Table IV

Involvement of Nerve

	Ulnar	Lat.Pop.	Total
Dapsone	20	8	28
Rifampicin	20	6	26
Total	40	14	54

Table V

Frequency of Investigations

No.of studies	No.of patients	No.of patients with initial abnormal NCV/EMG findings.
1	2	1
2	7	2
3	18	7
4	26	9
Total	53	19

Leprosy Patients : Nerve Conduction & E.M.G.Studies before & during Dapsone and Rifampicin Therapy

Table VI

(Patients with only one study)

Sr. No.	Reg.No./Name Age/Sex/Drug	Ulnar Nerve Motor				Ulnar Nerve Sensory				Lateral Pop.Nerve				E.M.G.	Remarks
		Rt.		Lt.		Rt.		Lt.		Rt.		Lt.			
		D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec	Lat. Msec	Amp. uv	Lat. Msec	Amp. uv	D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec		
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1	13768/G.L./17M/RFP	?.1	A53.3	2.0	61.8	ANo Resp.		1.94	15.0	-	-	-	-	Normal	Rt.Ulnar Neuropath
2	14982/N.N./25M/DDS	2.7	57.5	A2.8	50.7	2.3	30.0	A2.64	30.0	-	-	-	-	Normal	Normal

. D.L. = Distal Latency
. C.V. = Conduction Velocity
. M/Sec = Metres Per Second
. A = Clinically Thickened

Table - VII
(Patients in whom 2 studies done)

Sr. No.	Reg.No./Name Age/Sex/Diag	Ulnar Nerve Motor				Ulnar Nerve Sensory				Lateral Pop.Nerve				E.M.G.	Remarks
		Rt.		Lt.		Rt.		Lt.		Rt.		Lt.			
		D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec	Lat. Msec	Amp. uv	Lat. Msec	Amp. uv	D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec		
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1	9660/L.W./23M/RTF														
	I 22.2.86	-	-	-	-	-	-	-	-	4.58	48.7	4.32	A49.8	Normal	Normal
	II 8.11.86	-	-	-	-	-	-	-	-	4.6	53.3	3.72	A49.0	Normal	No chang
2	10318/B.W./20M/DDS														
	I 12.4.86	A2.44	46.7	2.42	62.3	A2.1	7.0	1.94	30.0	-	-	-	-		Rt.U. Neuropath
	II 26.7.86	A4.3	57.8	2.44	59.6	No Resp.		1.98	30.0	-	-	-	-	Rt.1st DI Reduc.	Motor con improved. Dec.Sen. cond.
3	11706/G.K.A./15M/RTP														
	I 12.7.86	1.9	62.8	2.0	A65.5	1.9	25.0	A2.0	25.0	-	-	-	-	Normal	Normal
	II 11.10.86	2.4	71.5	2.5	A67.0	2.08	25.9	A2.58	25.0	-	-	-	-	Normal	Normal
4	12171/C.M.H./40F/RTP														
	I 9.8.86	A2.24	56.8	2.16	56.0	A1.92	25.0	1.7	50.0	-	-	-	-	Normal	Normal
	II 20.11.86	A2.2	55.3	2.02	55.2	A1.9	30.0	1.8	40.0	-	-	-	-	Normal	No.sig. change

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
5	15452/N.B.S/28M/RP														
	I 7.2.87	A2.4	59.3	2.4	59.4	A2.04	25.0	2.16	25.0	-	-	-	-	1st.DI Normal	
	II 20.5.87	A2.1	68.0	2.0	61.0	A1.76	30.0	1.86	25.0	-	-	-	-	Normal	
														Normal	Normal
6	17345/B.C.T./38M/DS														
	I 9.7.87	A1.6	62.5	1.7	60.3	A1.52	40.0	1.74	40.0	-	-	-	-	Normal	Normal
	II 1.8.87	A1.74	63.0	2.0	61.3	A1.58	35.0	1.58	40.0	-	-	-	-	Normal	Normal
7	13962/B.C.T./40M/DS														
	I 25.7.87	A2.8	37.3	2.3	61.5	A	No Resp.	1.82	10.0	-	-	-	-	Rt.1st	Rt.Ulnar
														DI	Dec.Neuropath
														Lt.1st	
	II 24.11.87	A3.8	41.2	2.6	57.3	A	No Resp.	2.14	10.0	-	-	-	-	DI	
														Same	Same

Table - VIII
Patients with 3 studies

Sr. No.	Reg.No./Name Age/Sex/Drug	Ulnar Nerve Motor Rt. D.L. Msec	Ulnar Nerve Motor Rt. C.V. Msec	Ulnar Nerve Motor Lt. D.L. Msec	Ulnar Nerve Motor Lt. C.V. Msec	Ulnar Nerve Sensory Rt. Lat. Msec	Ulnar Nerve Sensory Rt. Amp. uv	Ulnar Nerve Sensory Lt. Lat. Msec	Ulnar Nerve Sensory Lt. Amp. uv	Lateral Pop.Nerve Rt. D.L. Msec	Lateral Pop.Nerve Rt. C.V. Msec	Lateral Pop.Nerve Lt. D.L. Msec	Lateral Pop.Nerve Lt. C.V. Msec	E.M.G.	Remarks
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
1	12655/B.S./20M/RFP														
	I 6.9.86	-	-	-	-	-	-	-	-	A3.96	45.0	3.76	44.3	Normal	Normal
	II 7.2.87	-	-	-	-	-	-	-	-	A5.32	43.3	4.3	43.3	Normal	Normal
	III 25.7.87	-	-	-	-	-	-	-	-	A3.4	46.4	3.6	46.5	Normal	Normal
2	12951/T.D./20M/DLJ														
	I 20.9.86	-	-	-	-	-	-	-	-	A4.0	46.0	3.7	46.5	Mild Redu. Rt.EDB	Rt.Lat.P Neuropath
	II 21.2.87	-	-	-	-	-	-	-	-	A5.1	42.8	4.3	41.0	Normal	Normal
	III 12.9.87	-	-	-	-	-	-	-	-	A3.82	46.0	4.2	52.8	Normal	Normal
3	13201/S.G./16F/RFP														
	I 4.10.86	2.7	65.0	A2.0	52.4	1.76	10.0	A2.0	20.0	-	-	-	-	Normal	Rt.Ulna Neuropath
	II 21.2.87	2.36	56.8	A3.3	55.8	1.92	20.0	A1.9	20.0	-	-	-	-	Normal	Improve
	III 9.4.87	2.02	59.07	A2.1	61.2	1.8	15.0	A1.8	20.0	-	-	-	-	Normal	Same as II

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
4	14306/V.H./21M/REP														
	I 6.12.86	-	-	-	-	-	-	-	-	5.4	47.7	A5.94	47.4	Normal	Normal
	II 18.4.87	-	-	-	-	-	-	-	-	4.34	56.2	A4.6	50.1	Normal	Normal
	III 21.11.87	-	-	-	-	-	-	-	-	6.2	54.5	A6.06	51.5	Normal	Normal
5 -	7635/P.D./20M/DDS														
	I 13.7.85	-	-	-	-	-	-	-	-	A3.4	48.3	4.12	51.0	Rt.EDL Dec.	Improve in interference patt.
	II 9.8.86	-	-	-	-	-	-	-	-	A3.36	50.2	3.7	52.2	Normal	Rt.EDB
	III 16.5.87	-	-	-	-	-	-	-	-	A4.14	52.6	3.7	49.5	Normal	Normal
6	9659/A.L./18F/DDS														
	I 22.2.86	1.68	50.3	1.5	A53.0	1.6	30.0	A1.7	50.0	-	-	-	-	Normal Cond. low but normal	Lt.U. Nerve limit
	II 5.7.86	1.9	62.8	1.7	A55.6	1.8	30.0	A1.5	30.0	-	-	-	-	Normal	No chan
	III 14.2.87	1.88	59.8	1.7	A53.3	1.6	25.0	A1.9	40.0	-	-	-	-	Normal	Normal
7	10320/D.M.W./35M/DDS														
	I 12.4.86	-	-	-	-	-	-	-	-	A6.42	46.7	3.88	50.8	Normal	Distal lat.Rt. side De
	II 30.8.86	-	-	-	-	-	-	-	-	A4.86	46.2	3.56	51.2	Normal	Improve
	III 27.6.87	-	-	-	-	-	-	-	-	A4.9	51.8	3.34	45.5	Normal	Normal
8	11927/A.T./21M/DDS														
	I 26.7.86	-	-	-	-	-	-	-	-	A5.9	47.6	4.7	47.0	Normal	Normal
	II 3.11.86	-	-	-	-	-	-	-	-	A4.8	46.3	3.9	45.3	Normal	No chang
	III 12.9.87	-	-	-	-	-	-	-	-	A4.9	47.0	3.6	47.5	Normal	Normal

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
9	12538/M.T./15M/DDS														
	I 30.3.86	2.08	59.0	A1.9	58.8	1.76	30.0	A1.88	40.0	-	-	-	-	Normal	Normal
	II 22.11.86	3.06	59.2	A2.82	53.2	2.74	25.0	A2.7	25.0	-	-	-	-	Normal	Normal
	III 23.5.87	2.2	61.5	A2.3	60.0	2.0	25.0	A2.0	25.0	-	-	-	-		Normal
10	15579/H.N./21M/DDS														
	I 14.2.87	2.3	55.4	A2.78	52.3	2.32	20.0	A2.4	25.0	-	-	-	-	Normal	Normal
	II 23.5.87	2.7	58.7	A2.74	55.8	2.06	25.0	A2.2	25.0	-	-	-	-	Normal	Normal
	III 9.1.88	2.6	61.8	A2.72	57.6	2.20	15.17	A2.1	15.17	-	-	-	-	Normal	Normal
11	1498/S.J.S./25M/RFP														
	I 10.1.87	-	-	-	-	-	-	-	-	A5.42	48.2	4.6	51.8	Normal	Normal
	II 9.5.87	-	-	-	-	-	-	-	-	A3.1	51.3	3.4	52.1	Normal	Normal
	III 16.1.88	-	-	-	-	-	-	-	-	A2.9	54.0	2.9	49.9	Normal	Normal
12	14034/D.Z.C./35M/RFP														
	I 20.11.86	A2.36	52.2	2.2	60.2	A	No Resp.	2.1	20.0	-	-	-	-	Normal	Rt.Ulna Neuropath
	II 21.3.87	A2.76	39.9	2.2	63.3	A	No Resp.	2.1	25.0	-	-	-	-	Normal	Deterio ration L
	III 29.8.87	A2.5	57.1	2.2	58.5	A	No Resp.	No Resp.		-	-	-	-	Normal	Improve ment Rt Deterio ration
13	14164/D.W./22M/DDS														
	I 29.11.86	A2.9	52.5	2.4	56.2	A2.5	10.0	2.3	25.0	-	-	-	-	Normal	Rt.U. Neuropath
	II 21.3.87	A	56.0	A2.3	58.8	2.0	10.0	A2.1	25.0	-	-	-	-	Normal	Same
	III 19.9.87	A2.3	63.0	2.1	64.0	A2.1	10.0	2.0	25.0	-	-	-	-	Normal	Same

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
14	14037/V.B./23M/DDS														
	I 22.11.36	2.7	63.0	A2.9	58.8	2.3	10.0	A2.2	25.0	-	-	-	-	Rt.1st DI	Rt.U. Neuropath
	II 14.3.37	A2.3	60.1	2.5	60.7	A2.0	22.0	2.08	25.0	-	-	-	-	Rt.sen.	Rt.Sen. Neuropath
	III 26.9.37	A2.3	64.5	2.7	59.2	A2.0	20.0	2.0	25.0	-	-	-	-	Normal	Improve
15	14501/J.P./35F/DDS														
	I 20.12.36	A2.78	48.2	2.8	54.8	A1.8	10.0	1.9	30.0	-	-	-	-	Normal	Rt.U. Neuropath
	II 28.3.37	A2.96	56.0	1.7	61.6	A1.9	10.0	1.8	35.0	-	-	-	-	Normal	Same
	III 12.9.37	A2.3	53.8	1.9	57.0	A1.8	10.0	1.84	30.0	-	-	-	-	Normal	Same
16	14044/F.Bee/23F/PTP														
	I 3.1.37	2.06	58.0	A2.1	55.2	1.8	30.0	A2.02	25.0	-	-	-	-	Normal	Normal
	II 18.4.37	1.86	61.8	A1.9	58.8	1.46	30.0	A1.7	25.0	-	-	-	-	Normal	Normal
	III 28.11.37	2.44	57.4	A1.98	49.3	2.0	30.0	A2.4	30.0	-	-	-	-	Normal	Normal
17	12656/V.K./38F/DDS														
	I 6.9.84	2.0	64.5	A2.5	62.5	1.9	25.0	A1.94	25.0	-	-	-	-	Normal	Normal
	II 20.12.84	2.6	63.0	A2.4	60.0	2.1	30.0	A2.12	30.0	-	-	-	-	Normal	No chan
	III 11.7.87	2.18	60.8	A1.7	65.3	1.8	35.0	A1.9	30.0	-	-	-	-	Normal	Normal
18	13071/RS/22M/DDS														
	I 27.9.86	A2.64	64.3	2.1	61.0	A2.0	25.0	2.0	25.0	-	-	-	-	Normal	Normal
	II 11.7.87	A2.3	58.3	2.2	60.0	A2.0	25.0	2.0	25.0	-	-	-	-	Normal	Normal
	III 21.2.88	A2.2	61.0	2.0	58.8	A2.0	25.0	2.0	25.0	-	-	-	-	Normal	Normal

Table IX

Patients with 4 studies

Sr. No.	Reg.No./Name Age/Sex/Drug	Ulnar Nerve Motor				Ulnar Nerve Sensory				Lateral Pop.Nerve				E.M.G.	Remarks
		Rt.		Lt.		Rt.		Lt.		Rt.		Lt.			
		D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec	Lat. Msec	Amp. uv	Lat. Msec	Amp. uv	D.L. Msec	C.V. Msec	D.L. Msec	C.V. Msec		
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15	16.
1	7612/P.G./71M/RFP														
	I 5.10.85	A2.14	61.7	2.06	58.0	A2.0	30.0	1.7	40.0	-	-	-	-	Normal	Normal
	II 8.2.86	A2.52	60.4	2.5	54.0	A2.28	30.0	2.28	50.0	-	-	-	-	Normal	Normal
	III 25.5.86	A1.98	64.8	1.94	62.0	A1.78	25.0	1.68	25.0	-	-	-	-	Normal	Normal
	IV 16.5.87	A1.92	65.0	1.8	63.5	A1.86	30.0	1.8	30.0	-	-	-	-	Normal	Normal
2	7614/T.K./16M/DDS														
	I 5.10.85	A2.44	59.3	2.5	57.0	A2.0	20.0	1.9	25.0	-	-	-	-	Normal	Normal
	II 8.2.86	A2.8	54.5	2.94	50.6	A2.2	25.0	2.4	30.0	-	-	-	-	Normal	Normal
	III 5.7.86	A2.5	62.8	2.84	63.4	A1.8	20.0	2.06	25.0	-	-	-	-	Normal	Normal
	IV 4.4.87	A2.6	68.0	2.54	59.0	A2.1	25.0	1.9	25.0	-	-	-	-	Normal	Normal
3	7753/M.P./36M/RFP														
	I 12.10.85	2.05	54.3	1.9	A59.0	1.4	20.0	A1.78	20.0	-	-	-	-	Normal	Normal
	II 19.4.86	2.20	58.8	2.3	A62.5	1.7	10.0	A1.98	40.0	-	-	-	-	Normal	Rt.U.Sen Neuro.
	III 27.9.86	A2.15	66.2	2.2	57.2	A1.4	25.0	1.84	25.0	-	-	-	-	Normal	Improved
	IV 25.4.87	A2.2	61.8	2.2	64.8	A1.6	20.0	1.9	35.0	-	-	-	-	Normal	Normal. No change from 1st study

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
4	9663/N.G./15F/RFP														
	I 15.6.85	A2.3	62.8	1.94	65.0	A1.9	10.0	1.7	25.0	-	-	-	-	Normal	Rt.U.S. Neuro.
	II 22.2.86	A2.1	66.7	2.02	67.0	A1.8	10.0	1.98	40.0	-	-	-	-	Normal	Unchange
	III 25.5.86	A1.9	60.5	1.78	62.0	A1.7	10.0	1.78	20.0	-	-	-	-	Normal	Lt.U.Ner Sens.Pot
	IV 10.6.87	A2.16	59.8	1.94	65.3	A1.6	15.20	1.82	25.0	-	-	-	-	Normal	Improved
5	7613/G.O.M./25F/RFP														
	I 5.10.85	2.14	60.0	A2.26	55.4	1.86	30.0	A1.78	50.0	-	-	-	-	Normal	Normal
	II 13.5.86	2.1	62.5	A1.94	60.7	1.7	45.0	A1.6	50.0	-	-	-	-	Normal	Normal
	III 13.12.86	2.36	58.3	A2.34	57.8	1.74	20.0	A1.92	25.0	-	-	-	-	Normal	Normal
	IV 4.7.87	2.0	62.8	A1.9	61.0	1.4	40.0	A1.5	40.0	-	-	-	-	Normal	No chang
6	7752/R.G.P./21M/RFP														
	I 12.10.85	-	-	-	-	-	-	-	-	A4.72	47.3	5.22	52.5	Rt.side EDB(Mild)	
	II 19.4.86	-	-	-	-	-	-	-	-	A4.2	46.6	4.43	52.0	Normal	Essen.un changed
	III 25.10.86	-	-	-	-	-	-	-	-	4.86	51.8	A4.2	46.6	Normal	
	IV 18.4.87	-	-	-	-	-	-	-	-	A4.52	54.0	4.6	54.3	Normal	Normal. unchange
7	8854/S.R/21F/RFP														
	I 12.12.85	2.0	55.3	A1.9	59.0	1.9	10.0	A1.9	12.0	4.2	53.8	A3.8	58.3	Normal	Done by. M.Baheti
	II 17.5.86	2.02	67.8	A2.1	59.2	1.56	40.0	A1.6	45.0	-	-	-	-	Normal	Essen. Normal
	III 6.12.86	1.94	62.8	A2.1	61.8	1.56	25.0	A1.94	25.0	-	-	-	-	Normal	
	IV 20.6.87	2.56	68.5	A1.8	58.0	1.5	40.0	A1.7	40.50	-	-	-	-	Normal	No chang

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
8	7637/K.M./25F/DDS														
	I 3.3.85	2.1	63.7	A2.0	55.6	1.9	15.0	A2.0	13.0	-	-	-	-	Normal	1st stud done by Dr.M.Bahe
	II 3.2.86	1.9	57.3	A2.5	62.3	1.8	30.0	A2.1	30.0	-	-	-	-	Normal	Normal N change
	III 9.8.86	1.5	60.3	A2.1	63.8	1.7	25.0	A1.7	25.0	-	-	-	-	Normal	Normal
	IV 16.5.87	1.3	65.0	A2.4	62.3	1.6	30.0	A1.9	30.0	-	-	-	-	Normal	No chang
9	9661/K.S.T./25F/RFP														
	I 22.1.86	A2.2	63.3	2.1	55.4	A1.7	37.0	2.1	50.0	-	-	-	-	Normal	
	II 17.5.86	A1.9	66.3	1.9	63.2	A1.5	25.0	1.5	40.0	-	-	-	-	Normal	Within Normal Limit
	III 3.1.87	A2.5	51.3	2.6	54.0	A2.3	25.0	2.6	25.0	-	-	-	-	Normal	Normal
	IV 11.7.87	A1.9	62.0	2.2	64.3	A1.6	25.30	1.8	30.0	-	-	-	-	Normal	Normal
10	11587/G.S.W./29M/RFP														
	I 5.7.86	2.1	63.0	A2.1	55.0	1.9	20.0	A1.8	17.0	-	-	-	-	Normal	U.Sen. Neuropat
	II 11.10.86	1.9	58.3	A2.2	56.0	2.1	20.0	A2.6	10.0	-	-	-	-	Lt.1st DI Mild	Lt.U.Neu Incre.
	III 9.5.87	2.2	60.5	A2.1	55.7	1.9	15.0	A2.1	12.0	-	-	-	-	Normal	Bil.Sen. Neuropat
	IV 2.1.88	3.1	55.0	A3.4	48.5	2.7	12.0	A	7.0	-	-	-	-	Normal	Deterio-ration
11	10906/S.T./17M/DDS														
	I 25.5.86	A2.0	55.8	1.9	60.0	A1.8	25.0	1.8	25.0	-	-	-	-	Normal	Normal
	II 30.8.86	A2.2	58.7	2.0	57.5	A1.6	40.0	1.9	40.0	-	-	-	-	Normal	Normal
	III 28.3.87	A2.1	62.5	2.0	59.0	A1.7	30.0	1.7	30.0	-	-	-	-	Normal	Normal
	IV 10.10.87	A2.2	58.8	2.2	59.5	A1.9	40.0	1.9	35.0	-	-	-	-	Normal	No chang

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
12	11321/V.N./35M/DDS														
	I 19.7.86	A2.3	64.2	A1.96	61.7	A1.94	25.0	A1.75	25.0	-	-	-	-	Normal	Normal
	II 4.10.86	A2.06	63.8	A1.9	60.0	A1.8	25.0	A2.16	25.0	-	-	-	-	Normal	No chang
	III 4.4.87	A2.2	66.5	A1.9	60.0	A1.7	25.0	A1.6	25.0	-	-	-	-	Normal	No chang
	IV 2.1.88	A2.2	57.2	A2.36	59.2	A2.0	20.0	A2.1	25.0	-	-	-	-	Normal	No chang
13	10310/N.G./22F														
	I 12.4.86	1.96	66.8	A1.93	61.3	1.54	20.0	A1.52	35.0	-	-	-	-	Normal	Normal
	II 9.9.86	/RFP36	63.7	A1.7	62.8	1.33	25.0	A1.42	25.0	-	-	-	-	Normal	Normal
	III 4.4.87	1.86	64.6	A1.84	63.2	1.6	20.0	A1.64	25.30	-	-	-	-	Normal	No chang
	IV 7.11.87	2.1	61.1	A2.1	59.5	1.73	20.0	A1.6	25.0	-	-	-	-	Normal	No chang
14	3564/P.D./40M/DDS														
	I 7.12.85	2.46	56.2	A2.54	54.5	2.04	40.0	A2.36	5.0	-	-	-	-	Normal	Lt.U.Sen Neuropath
	II 26.7.87	1.93	57.5	A2.3	61.3	1.8	30.0	A1.92	10.0	-	-	-	-	Normal	No sig. change
	III 7.2.87	2.3	54.3	A2.32	53.3	2.06	30.0	A2.2	5.0	-	-	-	-	Normal	No chang
	IV 18.7.87	2.14	56.3	A2.3	57.4	2.0	30.0	A2.0	5.0	-	-	-	-	Normal	No chang
15	9972/S.P./23M./RFP														
	I 19.4.86	2.32	54.2	A2.7	47.6	2.02	15.0	ANo Resp.		-	-	-	-	Normal	Lt.U.Sen Neu.
	II 26.7.86	2.4	60.2	A2.5	48.5	2.16	25.0	ANo Resp.		-	-	-	-	Normal	No sig. change
	III 14.3.87	2.5	55.7	A2.24	46.5		15.0	ANo Resp.		-	-	-	-	Normal	No chang
	IV 10.10.87	2.3	55.3	A2.3	46.3	2.38	25.0	A2.5	5.7	-	-	-	-	Normal	Mild Improvement Lt.U.Sen Nerve

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
18	8355/A.G./23M/RFP														
	I 23.12.85	2.7	56.6	A2.6	57.7	2.2	12.0	A2.1	18.0	-	-	-	-	Normal	Normal
	II 2.3.86	1.9	63.8	A2.06	60.2	1.9	30.0	A2.1	30.0	-	-	-	-	Normal	No sig. change
	III 23.3.87	2.2	58.5	A2.25	56.8	1.8	20.0	A2.08	25.0	-	-	-	-	Normal	Normal
	IV 25.9.87	2.1	67.2	A2.2	59.5	2.2	25.0	A2.2	25.0	-	-	-	-	Normal	No chang
17	12537/K.K./21M/RFP														
	I 30.3.86	-	-	-	-	-	-	-	-	A3.44	46.4	3.44	45.0	Normal	Normal
	II 29.11.86	-	-	-	-	-	-	-	-	A3.58	43.4	4.0	48.4	Normal	No chang
	III 23.5.87	-	-	-	-	-	-	-	-	A3.6	51.2	3.26	49.7	Normal	Normal
	IV 9.1.88	-	-	-	-	-	-	-	-	A4.16	49.8	3.7	49.5	Normal	Normal
16	10907/N.B.G./16F/RFP														
	I 24.5.86	A1.86	63.8	1.52	56.5	A1.6	25.0	1.7	25.0	-	-	-	-	Normal	Normal
	II 6.9.86	A1.76	64.3	1.9	64.5	A1.5	30.0	1.6	30.0	-	-	-	-	Normal	Normal
	III 25.4.87	A1.6	64.3	1.9	64.5	A1.5	25.0	1.5	30.0	-	-	-	-	Normal	Normal
	IV 31.10.87	A2.0	61.2	1.9	58.4	A1.6	25.0	1.7	20.0	-	-	-	-	Normal	Normal
19	11822/L.N./23F/DDS														
	I 19.7.86	-	-	-	-	-	-	-	-	A3.7	54.2	A2.9	51.3	Normal	Normal
	II 4.10.86	-	-	-	-	-	-	-	-	A3.3	49.5	A3.2	50.5	Normal	No sig. change
	III 4.4.87	-	-	-	-	-	-	-	-	A3.9	52.7	A2.6	49.5	Normal	Normal
	IV 2.1.88	-	-	-	-	-	-	-	-	A4.4	48.1	A4.0	47.8	Normal	No chang
20	11538/V.M.D./25M/DDS														
	I 5.7.86	A2.7	66.4	A2.3	60.0	A2.0	5.0	A2.0	20.0	A6.34	53.0	A4.16	52.3	Rt.EDB	Amp Rt.ED Rt.U & R Lat Pop. Neuro.
	II 20.9.86	A2.6	60.0	A2.3	60.2	ANo	Res	A2.1	25.0	A6.7	50.0	A3.66	51.0	Rt.EDB	Mild dete Rt.U.Neur
	III 4.4.87	A2.9	60.7	A2.2	60.5	ANo	Res	A2.1	25.0	A5.8	56.7	A3.4	52.3	Rt.EDB	Same
	IV 28.11.87	A2.3	46.2	A2.1	59.0	ANo	Res	A2.3	25.0	A4.1	45.2	A3.9	50.6	Rt.EDB	Deterio-ration R U.Neuro.

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
21	9973/M.S./24M/DDS														
	I 17.5.86	-	-	-	-	-	-	-	-	5.2	49.6	A4.7	49.6	Normal	Distal Laten.Rt side mor
	II 30.8.86	-	-	-	-	-	-	-	-	5.3	43.2	A4.1	40.4	Normal	No chang
	III 21.3.87	-	-	-	-	-	-	-	-	5.9	44.8	A4.5	43.2	Normal	Normal
	IV 29.3.87	-	-	-	-	-	-	-	-	6.3	41.0	A4.9	47.8	Normal	No chang
22	9459/T.M.P./21M/DDS														
	I 3.2.86	A2.3	52.9	A2.6	43.3	2.1	37.0	A2.2	25.0	-	-	-	-	Modera-tely redu.1st DI Lt.	Lt.U. Neuropath
	II 19.7.86	2.6	50.6	A2.2	51.2	1.9	35.0	A2.1	40.0	-	-	-	-	Normal	Improve-ment
	III 21.2.87	2.5	54.7	A3.0	34.5	2.1	25	A2.5	15.0	-	-	-	-	Lt.1st DI	Lt.U.Neu ropathy
	IV 3.3.87	2.4	62.6	A2.5	36.0	1.9	30	A2.6	7.10	-	-	-	-	Lt.1st DI	Deterio-ration
23	13322/J.G./16F/PEP														
	I 11.10.86	A1.7	64.2	2.0	58.0	A2.10	25.0	1.96	20.0	-	-	-	-	Normal	Normal
	II 14.2.87	A1.7	59.8	2.2	55.4	A1.3	25.0	1.9	10.0	-	-	-	-	Normal	Lt.Ul. Sensory
	III 8.3.87	A1.9	56.5	2.1	56.3	A2.0	25.0	2.09	12.14	-	-	-	-	Normal	Same
	IV 13.2.88	1.82	50.8	2.1	62.0	1.84	20.0	2.0	25	-	-	-	-	Normal	Normal
24	8563/P.K./17M/KPP														
	I	2.18	66.3	A2.0	62.0	1.88	30.0	A2.06	20.0	-	-	-	-	Normal	Normal
	II 17.12.85	2.18	67.0	A2.38	60.0	1.9	25.0	A2.14	20.0	-	-	-	-	Normal	Normal
	III 12.4.86	2.0	64.5	A1.9	63.3	1.6	25.0	A1.86	20.0	-	-	-	-	Normal	Normal
	IV 26.6.87	2.1	60.2	A2.36	58.3	2.2	40.0	A2.18	30.0	-	-	-	-	Normal	Normal

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.	15.	16.
25	11705/S.B./13M/RFP														
	I 12.7.36	-	-	-	-	-	-	-	-	No	Resp.	A4.2	40.8	Rt.EDB	Rt.Lat.P
														Dec.	Neuropathy
	II 25.10.36	-	-	-	-	-	-	-	-	5.1	50.5	A3.9	50.0	Normal	Improved
	III 18.6.37	-	-	-	-	-	-	-	-	4.7	49.3	A3.3	52.5	Normal	Normal
	IV 30.1.38	-	-	-	-	-	-	-	-	4.3	49.2	6.0	52.0	Normal	Normal
26	11707/S.B./23M/DDS														
	I 12.7.36	2.2	55.3	2.66	59.3	3.05	7.00	1.9	20.0	-	-	-	-	Normal	Rt.U.
															Neuropathy
	II 25.10.36	2.4	50.5	2.42	58.0	2.1	17.0	2.14	20.0	-	-	-	-	Normal	Improved
															in sen.
	III 20.6.37	2.26	50.0	2.6	59.3	2.0	25.0	2.16	25.0						conductio
	IV 5.2.38	2.54	55.4	2.94	60.5	2.1	17.20	2.4	25.0	-	-	-	-	Normal	Improved
														Rt.Sen.	
														Pot.Dec.	
														Improv.	

TABLE X

Clinical and Electroneuro Physiological correlation

Sr. No.	Regd. No.	Name/age sex/ready	Patches	Nerve thick.	Peripheral Anesthesia Hlthy.	Aff.	Motor Deficit Hlthy.	Aff.	Sensory cond. Amplitude Hlthy.	Aff.	Sensory cond. Latency Hlthy.	Aff.
1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
1	65	Ratan/22M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
2	53	Bandu/21M										
		Initial	A	T	Ab	P	Ab	P	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
3	61	Tulsidas/20M										
		Initial	A	T	P	Ab	Ab	Ab	N	Mild Reduct. EDB	N	Neuro-pathy
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
4	64	Sharda/16F										
		Initial	A	T	Ab	Ab	Ab	Ab	N	Reduced	N	Reduced
		Final	A	T	Ab	Ab	Ab	Ab	N	N	N	N

1.	2.	3.		4.	5.	6.	7.				
5	66	Jyoti/15F									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
6	71	Vithal/16M									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
7	37	Prakash/20M									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
8	39	Pramod/17M									
		Initial	A	T	Ab	P	Ab	P	N	N	N	N
		Final	NP	T	Ab	Ab	Ab	Ab	N	N	N	N
9	46	Aruna/17F									
		Initial	A	TT	Ab	Ab	Ab	Ab	N	N	N	N
		Final	NP	N	Ab	Ab	Ab	Ab	N	N	N	N
10	50	Dasharath/35M										
		Initial	A	T	Ab	P	Ab	P	-	-	N	Increased
		Final	F	T	Ab	Ab	Ab	Ab	-	-	N	N

1.	2.	3.	4.	5.	6.	7.						
11	52	Baban/15M									M	No Resp.
		Initial	A	T	Ab	Ab	Ab	Ab	-	-		
		Final	F	T	Ab	Ab	Ab	Ab	-	-	N	N
12	19	Prasad/22M									In	In
		Initial	A	N	Ab	Ab	Ab	Ab	D	D		
		Final	F	N	Ab	Ab	Ab	Ab	N	N	I	N
13	56	Ashok/25M									N	N
		Initial	A	T	Ab	P	Ab	P	N	N		
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
14	49	Mahendra/16M									N	N
		Initial	A	TT	Ab	Ab	Ab	Ab	N	N		
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
15	78	Maroti/21M									N	N
		Initial	A	T	Ab	Ab	Ab	Ab	N	N		
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
16	74	Suryabhan/30M									N	N
		Initial	A	T	Ab	Ab	Ab	Ab	-	-		
		Final	F	T	Ab	Ab	Ab	Ab	-	-	N	N

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
17	69	Dhyaneshwar/35M										
		Initial	A	T	Ab	P	Ab	P	N	No Resp.	N	No Resp.
		Final	F	T	Ab	Ab	Ab	Ab	Det.	Det.	Det.	Det.
18	70	Deepak/22M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	Low	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	Low	N	N
19	63	Vithal/28M										
		Initial	A	T	Ab	P	Ab	P	N	Low	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
20	73	Janabai/39F										
		Initial	A	T	Ab	Ab	Ab	Ab	N	Low	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	Low	N	N
21	76	Fatimabi/33F										
		Initial	A	T	Ab	P	Ab	P	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
22	62	Vatsala/35F										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
23	40	Prakash/21M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	IIF	T	Ab	Ab	Ab	Ab	N	N	N	N
24	42	Kirtam/15M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
25	38	Bharati/35M										
		Initial	B	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
26	36	Manda/16F										
		Initial	B	TT	Ab	Ab	Ab	P	N	Low	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
27	41	Girja/29F										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
23	32	Ramesh/22M										
		Initial	A	TT	Ab	P	Ab	P	-	-	N	N
		Final	F	N	Ab	Ab	Ab	Ab	-	-	N	N

1.	2.	3.		4.	5.	6.	7.	8.			
29	35	Shakuntala/25F									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
30	38	Kalawati/23F									
		Initial	C	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	T	Ab	Ab	Ab	Ab	N	N	N	N
31	44	Kamal/25F									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
32	58	Gangadhar/26M									
		Initial	A	T	Ab	Ab	Ab	Ab	N	Low	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	Low	N	N
33	55	Suresh/17M									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
34	9	Vasant/33M									
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
35	43	Kunda/20F										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
36	13	PL.Dalpe/39M										
		Initial	A	TT	Ab	Ab	Ab	Ab	N	Low	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
37	7	Pathan/27M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	No Resp.	N	No Resp.
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
38	29	Adinath/26M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N
39	60	Kisna/21M										
		Initial	A	T	Ab	Ab	Ab	Ab	-	-	N	N
		Final	F	N	Ab	Ab	Ab	Ab	-	-	N	N
40	53	Nirmala/16M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	N	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N

1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
41	10	Laxmi/23F										
		Initial	A	T	Ab	Ab	Ab	Ab	-	-	N	N
		Final	F	I	Ab	Ab	Ab	Ab	-	-	N	N
42	14	Vijay/21M										
		Initial	B	[illegible]	-	P	Ab	Ab	-	Low	N	N
		Final	F	N	-	Ab	Ab	Ab	-	N	N	N
43	46	Madhukar/27M										
		Initial	A	T	Ab	Ab	Ab	Ab	-	-	N	N
		Final	F	N	Ab	Ab	Ab	Ab	-	-	N	N
44	45	Patil/21M										
		Initial	A	T	Ab	Ab	Ab	Ab	N	Low	N	N
		Final	F	N	Ab	Ab	Ab	Ab	N	N	N	N

ABREVIATION

A. No.of patches limited to 1 to 3.
B. No.of patches limited to 3 to 5

C. No.of patches more than 5

N. No thickening of nerve
T. Nerve thickening present
TT. Nerve thick and tender

P. Present
Ab. Absent
F. Faint
NP. No patch
D. Decrease
In. Increase
Det. Deterioration
Hlthy. Healthy
Aff. Affected

Table XI

Association between peripheral anesthesia and sensory conduction studies

erve	No.of Nerves investi-gated	Peripheral Anesthesia	Sen.Latency		Sen.Amplitude	
			Normal	Abnormal	Normal	Abnormal
ealthy						
	--	Present	--	--	--	--
	32	Absent	31	1	31	1
ffected						
	5	Present	3	2	3	2
	27	Absent	24	3	19	8

Table XII

Correlation between No. of patches and sensory conduction studies

No.of Patches	Nerve Status	Sensory Latency		Sensory Amplitude	
		Normal	Abnormal	Normal	Abnormal
1-2					
	Healthy	28	-	28	-
	Affected	24	4	20	8
3-5					
	Healthy	2	1	2	1
	Affected	2	1	1	2
> 5					
	Healthy	1	-	1	-
	Affected	1	-	1	-

TABLE XIII-a

ELECTRONEURO PHYSIOLOGICAL STUDIES

	MNCV(Ulnar)		MNCV(Lat.Pop.)	
	Healthy	Affected	Healthy	Affected
Dapsone				
Initial	53.61+3.16	55.06+7.61	43.98+2.22	49.39+2.94
Final	59.76+3.6	56.07+9.07	41.9	47.9 +1.913
Rifampicin				
Initial	59.66+4.04	58.43+4.36	42.73+17.54	46.64+2.74
Final	62.42+3.80	57.80+6.01	51.17+3.15	51.93+2.10

TABLE XIII-b

ELECTRONEURO PHYSIOLOGICAL STUDIES

	Distal latency (u)		Distal latency (l.p.)	
	Healthy	Affected	Healthy	Affected
Dapsone				
Initial	2.30+.34	2.42+0.39	4.52+0.01	4.61+1.31
Final	2.37+0.363	2.34+0.157	6.3+	4.68+0.854
Rifampicin				
Initial	2.13+0.264	2.14+0.249	3.95+1.73	4.68+0.354
Final	2.18+0.335	2.16+0.45	4.2+0.453	4.39+0.975

TABLE XIII-c

ELECTRONEURO PHYSIOLOGICAL STUDIES

	Sensory Amplitude		Sensory Latency	
	Healthy	Affected	Healthy	Affected
Dapsone				
Initial	24.9+9.33	21.25+13.0	1.96+0.35	1.93+0.5
Final	26.51+5.74	21.3+10.9	1.94+0.21	2.046
Rifampicin				
Initial	24.61+11.16	21.14+12.3	1.36+0.02	1.529+0.10
Final	27.93+3.77	24.93+11.29	1.93+0.3	1.885+0.33

ANIMAL EXPERIMENTS

It was proposed to carry out Animal experiments to find out the toxic effects of Dapsone, to test whether Dapsone has any toxic effect on the normal healthy nerves of the animals. There was experimental evidence of neuroprotective effect of Dapsone in leprosy infested nerves in mice (Kamala et al 1984). Despite this experimental animal, it was decided to use a bigger animal, in which infection with M. leprae is not possible, but the possible toxicity on normal nerves with larger and different doses can be tried. Further, a bigger animal would be more convenient to study nerve conduction velocity. The dog was chosen for the purpose.

Material methods:

Three groups of dogs were selected.

Group I : Dapsone to be given orally in suitable doses to produce in blood the same concentration as that leprosy patients reach on consumption of 100 mgs daily.

Group II : Dapsone to being given orally at 5 times the above dose.

Group III : Control group without Dapsone administration.

The blood concentration in man with an oral daily dose of 100 mg Dapsone was estimated by Marshall and Bratton method. Then starting from 25 mg repeated estimations of Dapsone were carried out in dogs. The dose which can produce blood level equavalent to that of human was calculated. It was found that 2 mg/kg of Dapsone in dogs can produce the blood level of Dapsone equivalent to that of human consuming 100 mg daily. So each dog was weighed repeatedly and corresponding dose of Dapsone was given orally, mixed with food. All the dogs were immunised against Rabies. All the contacts were immunised against tetanus. An animal house was improvised. The dogs were kept in cages. These cages were designed in such a way that each dog can move sepeately and freely inside the cage.

Follow up:

All the groups of dogs were initially subjected to nutritional assessment and nutritional deficiencies were corrected and balanced. The dogs were examined regularly at intervals of 7 to 10 days for any observative change in gait or weakness in the limbs or any other abnormality. An initial baseline recording of motor nerve conduction was recorded and at the end of every 3 months, motor nerve conduction velocity studies were carried out. The conduction studies are carried out

after anesthetising the dogs with ethyl chloride in a special chamber.

Observations and discussion:

There are 3 dogs one in each group, The conduction velocities in these three groups at regular intervals are as follows. Ulnar nerves are studied through out the follow up period.

Group I : The dog was fed with 2mg/kg dose of Dpsone

MNCV(metres/sec.)	Rt	Lt
Initial	43.2	45.0
3 months	40.3	42.0
6 months	35.23	36.84
9 months	42.3	46.5
12 months	44.4	53.88

Group II : Dog was fed with Dapsone 5 times that of human dose (Dapsone 10mg/kg)

MNCV (Meters/Sec)	Rt	Lt
Initial	30.0	36.0
3 months	28.0	38.0
6 months	20.0	33.5
12 months	25.0	30.3

Group III : Dog was ot fed with Dapsone.

MNCV (Meters/Sec)	Rt	Lt
Initial	36.0	38.2
3 months	30.0	32.0
6 months	39.5	38.0

As can be seen from the above observations group I, the dog receiving Dapsone in human concentration did not show any signifiant change either clinically or electro-physiologically. But group II, the dog receiving Dapsone in a dose of 10 mg/kg showed deterioration in the motor nerve conduction velocity. But clinically there is no development of deformity. The control group did not show any change either clinical or electro-physiological.

CONCLUSIONS

Retrospective Study :

A retrospective study has some inherent disadvantages. Precise information is not generally available as one has to depend on recorded data. Recording is not always uniform, since the personnel would have changed over the period. Giving a latitude to all these factors, in the present study it was grossly observed that a larger proportion developed deformity among the patients who were regular in treatment than among those who were less regular.

However in a group of patients who have consumed a fixed amount of drug (eg.33gm), more patients developed deformities in the group that consumed the drug in a longer period (2-5 years) than among those that consumed the same amount of the drug in a shorter period (upto 2 years).

Prospective Study:

In leprosy, no two cases are identical. Hence in this study an affected nerve and an apparently normal nerve in the same patient are studied to compare the possible damage to normal and infected nerve by dapsone therapy. Motor conduction velocity showed no significant difference between the affected and unaffected nerves. A number of possible causes for such a finding have been ennumerated. On the other hand, sensory amplitude and

sensory latency showed a significant difference between healthy and affected nerves. Further, sensory conduction changes could be elicited before clinically detectable anaesthesia. This was evident by the fact that in 59 cases without anaesthesia of limbs, there was diminished sensory amplitude in 5 cases and increased latency in 3 cases.

Following the patients on treatment, in present study there was no extension of anaesthesia or dimmunition of motor power. Electro physiological study did not show any significant difference between the initial and final recordings of motor and sensory nerve conductions if aggregate figures are taken into consideration. Taking individual cases, deterioration in nerve conduction was found only in 2 cases. In all the others there was no deterioration; and if there was a temporary deterioration, it improved or remained stationary with treatment and continuation of treatment did not cause any further damage. Of the 2 cases that showed deterioration, one was on Dapsone therapy and the other was on R.F.P.. It would not, therefore, be possible to incrimate DDS as the cause of nerve damage, and other factors involved in the disease process are to be considered.